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THE CONCEPT OF CARDIAC FAILURE
FROM HIPPOCRATES TO WHITE.

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Presented to the faculty of the University
of Nebraska College of Medicine in partial
fulfillment of the work for the degree of
Doctor of Medicine.

University of Nebraska
College of Medicine

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From the first dawn when Nature's extravagance evolved the human, he has stood in awe at Her power and secrecy. Also, since the first breath from eternity was blown into the breast of man, he has wondered how and why he was created - so like and yet so different than things below and above him.

It strikes us also, with that first breath was created the desire in that striking singularity, his mind, to solve Nature's secret of birth, life and death. And so science and more particularly medicine, has been born a child of man's wondering. (147)

We have no record of the first human to start the observations and writing of the facts which have led to our present understanding of why and how "today we live, tomorrow we are like trodden chaff from the threshing floor." (147)

It is probable, however, that long before our records show, men saw their fellows live and die and that when they failed to live, the breathing and the throbbing of their vessels had ceased and the "vital spirit" was gone. (58) So in a sense, the clinical picture of cardiac failure has been known to man always. The riddle presented to the ancient is still little understood. (136)

In all mortality tables there is one cause which mounts above the others - everywhere it is the principal cause of death. (3)

The tendency is to associate preventive medicine with parasitic diseases "but preventive medicine must fight one of its greatest battles within the pericardium." (147) Organic heart disease kills more human beings than any other condition. "Men's effort to solve this riddle stretches across the centuries from Aristotle to Einthoven and no chapter in medical history is more important than the development of our knowledge of the heart." (147)

It may be realized that with the problem ever before the cardiologist of prevention of cardiac failure yet without the subject being mentioned as a clinical entity, at least until very recent years, it becomes a difficult task to separate from the voluminous writings on the heart the time when the subject of "cardiac failure" crept into the minds of men and consequently into the literature. Or is it an entity yet today? Have we been able up to the present time to extract from the apparently frugal attempts of the cardiologist any definite means of telling why the heart, as Hippocrates called it, "the fountain of life" (136) should ever fail with its marvelous mechanism for compensation and response to overwork.

It is striking that the modern physiologist might not by now have offered us something in explanation of the fact that severe strain to the heart does not affect it in the same way that a severe stretching affects a skeletal muscle. Skeletal muscle, pathologists tell us heals by scar tissue formation yet autopsy reports show only occasional scarring in hearts subjected to the highest degree of overwork. (7 - 11)

How does it happen that with cardiac failure before medical men from the beginning of history and with a clinical picture so characteristic, that we find little reference to it as an entity in itself? (45) Does the heart itself ever fail? Is it myogenic, neurogenic, or the combination of many things thrown into the picture to keep the proper "ebb and flow" of the blood so necessary to the organism? Is the heart the marathon actor which must keep pace with intricacies in the functioning mechanism of an entire vascular system? Or is there still a more elusive undiscovered factor? (3)

It is not the writer's desire to determine the answer nor solve the ancient riddle of the heart. Rather we shall only consider the development of the concept of the almost mystic subject of "Cardiac decompensation" or perhaps more generally better known today as "cardio-vascular failure" or "cardiac insufficiency." The romantic development of

the subject takes us to the histories and development of all the medical sciences; in fact, in one sense to history at large. That is in the sense that cardiology has drawn from the history of science at large for its concept and practice.

Since the conception of the subject with which we are dealing has kept pace with the facts which have been discovered about the heart, it appears altogether fitting that we should consider the history and the manner in which step by step, we have gained our knowledge of modern cardiology. The history of cardiology has been considered by Faugers and Bishop as having three periods of development; (58) the pre-scientific, the scientific and the period of scientific application.

The pre-scientific period was developed by men who were limited, one might say "almost imprisoned by circumstances of their times." They were almost entirely unable to apply the knowledge they did have because of the mechanical imperfections of the methods employed in their investigations. (58) Would it not be a marvelous thing if one endowed with such a philosophical mind as Hippocrates might be reincarnated and devote himself to the study of modern medicine with all the modern aids present to ~~help~~ him in his experimental and clinical studies? (58) We should probably witness even more

astonishing discoveries than any of our forefathers have yet produced.

A word of wisdom, it appears, has been written by Normal W. Thiesen as he viewed the development of humoral medicine. "Today in our ignorance we conceive that at last the final word has been said regarding the functions of the human body. Such papers are prepared and we complacently smile as we read our of our ancestors believing in such things as melancholy humors and the circulation of the vital spirit. Yet, when one reads in modern journals of auto-intoxication, the x factor in the causation of ulcer, tissue death and animal emanations, one is inclined to more and more uncertainty in the belief that we are living in a medical Utopia. The diadactic teachings of today may tomorrow be regarded as the evanescent babblings of a theoretic dogmatism." (147)

So it may be with our subject at hand. Are we any farther than the Hippocritic belief during the first period that the objective evidences of circulation and its disorders were considered with awe and were manifestations of some supernatural influence acting upon the human body. (163) It was not until after the fifth century that men began to emerge from the binding veil of philosophical and religious influences. In ancient Egypt, which is considered the "cradle of civilization", very little was

known about the heart in a scientific way. (147)

We are at once presented with the fact that cardiology is not dependent upon a people but upon all of civilization for its scientific growth. (58) It has kept pace with the human race in general. We are also aware of the fact, from recent examinations of mummies, according to Faugers and Bishop, that the ancients were affected with many of the diseases of the vascular system so well known to us such as arteriosclerosis.

Considering some of the men of the pre-scientific period, our attention is drawn first to Plato, 427-347 B.C., who considered the heart in a philosophical sense and said it guarded the higher emotions. We find it mentioned by Alcmaeon in 384 B.C. who said that "sleep and death were connected with the circulation of the blood."

In 384 B.C. Aristotle lived. It was during this time that investigations were being worked out in a more complete fashion than ever. His contributions were mainly an attempt to show the functions of each organ. He studied the heart and the vessels and his belief was that the blood expanded in the vessels of the heart. He did not consider the pulmonary veins and arteries as a part of the circulatory system. He believed that the heart was all-important, was the center and contained only three cavities.

Praxagoras in 300 B. C. discovered the difference between the arteries and veins but believed the veins to be the main vessels.

Erastratus, 331 B.C., is said to be the originator of dissection. He studied the heart and believed the arterial and venous blood had an anastomosis which was called into play only during disease.

Hippocrates in 340-460 B.C. noted that people who have had frequent attacks of swooning without any manifest cause, die suddenly.

Although we notice that "inspectum cadaverum" was not practiced extensively by the ancients, we find it mentioned in many places. It is remarkable to read that during this time sudden deaths were recognized as being due to heart disease. (136) Theophilus, in his Commentary, mentions that Galen inferred this from finding disease in inferior hearts that had ceased beating suddenly.

A treatise upon the heart, which is not generally attributed to be original with Hippocrates, but of great antiquity, describes the auricles and the ventricles and the origin of the veins from the auricles. They further say that the orifice of the heart was the "fountain head" from which all parts of the body were irrigated. They believed the blood was not the irrigating substance but

rather "a pure, luminous superfluity from the heart."

(163)

It was not until 131 A.D. that Galen discovered that the arteries contained blood and not air, that we began to associate the heart with the blood. He further concluded that since the heart would beat outside the body, that the impulse must originate within itself. It was at this time we began to realize the importance of the heart muscle in relation to the circulatory system and then began to try to classify the variations in the pulse.

In 1250 A.D. Mondino DeLuzzi wrote a book "Anatomia" which, although full of errors and based on Arabic traditions, was the only anatomy for over a century. He doubted Galen's view of permeability of the septum. (58)

One of the earliest writings of the conception of "cardiac decompensation" is perhaps found in the description of the symptoms of patients who undoubtedly were cardiacs.

From Thiesen's writings of "Bartholemus Anglicus" we quote: "The first, or diaphoretic form of cardiacal affection, "heart passion" or "heart quaking", is due to heat and distemperance of heat above the spiritual members, that is the lungs. This brings an opening of the pores of the heart and the humors and vapors in the blood, which are resolved and shed. This fumosity finds its way through the pores

and is made thick and turns into sweat and from such frequent sweating of blood comes dropsy or hectic fever. Patients often swoon from defective strength (fainting from the heart.)" (147)

It is interesting to observe their conception of the old symptom of palpitation. "Tremens comes generally from melancholy, black bile or some times phlegmatic; for superfluities of such humors in the lungs press and wring, often either chamber of the heart as well as the substance thereof. And so the movements of the heart are interfered with and the veins are stopped (blocked) through which the spirits of life shall pass and these are suddenly stuffed (shut off.) Sometimes this affection comes from defect of the liver causing venous stasis. Thus failing to send sufficient feeding to the heart, as Constantine says "the heart faileth." (147)

"Sometimes the heart conditions come from a diseased disposition of the constitution, or from other parts of the body and the heart suffers by company thereof. (Sympathetically) This happens especially when running fluid and vapors or humors of a grievous nature come from the brain or from the stomach. By this substance the heart is hurt or grieved (affected) and fails. " (147)

It is in the above that we have, long ago, a suggestion of "heart failure." In another writing we see that the symptom of dyspnea was well known and associated with heart affectations.

"In these cases compressing and wringing of the heart (that is, interferred with its action), may come from the violence of fevers and from this there follows dryness and thirst, loud breathing, for it requires an effort to draw in the breath. The air from the heart may not open itself."
(136)

We pass along to the discoveries of Leonardo Davinici, 1452-1519. He gave an accurate account of the heart valves. He further believed that systole and not diastole was the important phase in the cycle of the heart.

Sylvius described the foramen ovale. Vesalius, 1514-1564, was heralded as the prominent anatomist until the time of Harvey. He is called the "Father of Anatomy." He still believed the auriculations to be separated. Vesalius was a martyr and was exiled for having been accused falsely of opening the body of a living man.

It may be here noted that although we are unable to find the specific subject "cardiac decompensation" or nosologic description of the subject as an entity in the early writings, it seems certain the authors knew that the heart failed. Perhaps they also recognized the clinical features but were unable to interpret them just as we are unable to entirely interpret them all today.

As we pass to the time of Servetus, 1509-52, we find another man, a martyr, who described the pulmonary circu-

lation as being separate from the main or systemic circulation. He was burned at the stake by Calvin for a writing on heresy.

In 1478-1522 we find Brissart re-employing, contrary to the belief of his time, a therapeutic measure introduced by Hippocrates for the relief of an "embarrassed circulation." (163) Hippocrates, in contrast to the "revulsive" bleeding of Brissart's time, bled on the same side of the lesion and near to it. (163) Brissart re-introduced this practice to the strong objection of the followers of the Arabic teachings. (58)

One of the greatest contributions to anatomy was made by William Harvey, 1587-1657. He discovered the circulation. In his "Mortu Cardis" (1628) he said that "the actual quantity of the blood as calculated by him made it physically impossible for it to do otherwise than return to the heart by the veins. The blood travels in a circle." (58)

Marcello Malphigi, 1628-94, founder of descriptive embryology, demonstrated the capillary circulation in the mesentery of a frog.

Giovanni Alfonso Borelli, 1608-79, suggested the neurogenic theory of the heart beat.

Niels Stensen described the true muscular tissue of the heart as the prominent factor in the heart beat.

John Mayon, 1643, stated that "dark red blood of the veins changed to bright red after combining with an element in the air." (58)

We are indebted to Francis Glisson, 1597, for proving that a contracting muscle does not increase in volume.

Giovanni Maria Lancisci, 1655-1720, described the cervical veins and noted for the first time the important pathological findings of hypertrophy and dilatation. He intimated further that the latter sometimes caused sudden death. He also attempted a classification of cardiac diseases, described valvular vegetations and observed aneurysms and syphilitic hearts. It was left to Giovanni Morgagni, 1682, to describe the first heart block.

Stephen Hales in 1677 first accurately measured blood pressure and Albrecht von Haller proposed the myogenic theory of the heart beat and also established Glisson's theory of the irritability of the muscle reaching it by the brain. (58)

Jean Baptiste Sennac, 1693-1770, is perhaps the first to actually publish a book on the structure of the heart, its action and diseases. He carried out in rather a methodical way, an enumeration of lesions and symptoms. He also studied irregularities of the heart to some extent and discussed such symptoms as orthopnea, dyspnea, and dropsy in relation to the heart. (58) He was perhaps the first to mention the fact, which has since been the unex-

pansive gap between the pathologist and the clinician -
"One cannot always connect the symptoms and signs in the living with the pathological findings in the cadaver.

It was about this time when the separate branches of medicine began the individualistic investigation of its different fields. "We began that seeming ever widening difference; though truly the 'main spring' in the disproof of dogmatic theories, which had been the experimental stigmata for years." (103)

It is not difficult to see that the imperical manner in which the ancients collected most of their data was a necessity. (58) "Cardiac decompensation" or "heart failure" we see, up to this time, was hardly considered. There were too many things to distract their attentions besides the end result of a wornout machine.

Until the time of Sennac medical men were struggling with the stifling influence of men who believed science a heresy. Their minds were clouded with the dogma of a purely phylisophical trend of thought. (58) It was not until we began to enter upon the third period, or the period of scientific application, according to Faugers and Bishop, that cardiology benefited from the knowledge so costly and laboriously collected by the ancients and men of the sixteenth and seventeenth centuries. He further states that it cannot be denied that the application of scientific

methods to the studies of the diseases of the heart has been of vast importance and its development, for the most part, has been dependent upon the discovery of mechanical instruments designed to aid clinical investigators. Experiments could be performed on a large scale with considerable degree of accuracy and properly controlled by known normal factors. (58)

Many names appear during this era. "Some of them stand out, as it were, like milestones on the road to the full development of cardiology." (58)

Leopold Auenbrugger, 1722-1809, first adopted the principle of physical acoustics. Jean Nicholas Couvisart, 1755, was the first to call himself a cardiologist. He made a statement which is most significant to the modern cardiologist as related to "cardiac decompensation." "Upon the muscular efficiency of the heart depends life itself." Another significant thing was his belief that hypertrophy and dilatation were the chief causes of abnormal cardiac function and that valvular disease was purely secondary to this.

Although he neglected the use of digitalis, William Withering, 1741, used ^{it} in a very modern fashion and was the first to use it in the proper dosage. He noted that it would produce amelioration of symptoms in patients suffering from edema and heart affections. He made no distinction

between cardiac and renal dropsy. We find no mention of the term "cardiac insufficiency", "heart failure" or any of the other terms relative to the failing heart in the literature up to this time. It is necessary then to consider the conceptions of medical men of the various symptoms associated with the condition which we have shown were known even to the Hippocratic period.

Digitalis, rather accidentally discovered, offered, as it were, a wedge to the opening of a consideration of what might be done for the failing heart and the relief of the objective symptoms so baffling to medical men. Although Withering was interested in the idea of dropsy, he was very observing in that he discovered the effect of digitalis upon the heart. (147) He knew there were different types of congestion and failure of the heart as shown by his use of digitalis in selected cases. To quote him: "If the pulse be feeble or intermitting, the countenance pale, the lips livid, the skin cold, the swollen belly soft and fluctuating and the anasarcaous limbs readily pitting under pressure of the finger, we may expect the diuretic effect of digitalis to follow in a kindly manner." We may determine his conception of the situation by quoting a case history prepared by him: "I found him supported upright in his chair by pillows, every attempt to lean back or stoop forward giving

him a sensation of instantaneous suffocation. He said he had not been in bed for many weeks. His countenance was sunk and pale; his lips livid, his belly, thighs and legs very greatly swollen; hands and feet cold, the nails almost black, pulse 160 tremulous beats in a minute but the pulsation in the carotid arteries was such as to be visible to the eye and to shake his head so that he could not hold it still. His thirst was very great. His urine scanty and he was disposed to purge."

"I immediately ordered a spoonful of infusion of digitalis every six hours, with a small amount of laudanum to prevent its running off by stool, and a decoction of leontodon taraxacum to allay his thirst.

"The next day he began to make water freely and could allow of being put to bed, but was raised high with pillows. I omitted the infusion that night and he parted with six quarts of water and the next night could lie down and sleep comfortably."

He tells of continuing his treatment until the swelling was all gone and the man returned home in a month.

Dawson and Dixon state that Withering recognized the action of digitalis upon the heart in his statement "that it has power over the motions of the heart to a degree yet unobserved in any other medicine, and that this power may be converted to salutary ends." Also in the remark "that

the pulse might be retarded to an alarming degree without any other preceeding effect." (146)

Another important advancement was accomplished when Jean Poisenille invented the mercury manometer and blood pressures were studied in relation to efficiency of the heart. (59) The invention of the sphygmograph by Morey in 1830 was not without its effect in the studies in the variation of the pulse. Thus we see that the advent of special instruments of experimentation and the advent of the drug digitalis in a sense opened the field for more scrutinizing study than ever before. (59)

In Billings time they were presented with the seemingly endless job of separating the symptoms caused by actual lung pathology and disease of the heart. They were confronted with the same distressing symptoms of decompensation, as medical men had always been, but scientific methods had not yet been developed to the extent that the real significance could be determined. (25) Billings endeavored to explain the meaning of "spasmodic" asthma and was well aware of the fact that the valvular diseases lead eventually to the failure of the heart. He further did not believe, as previously, that asthmatic conditions were caused by a contraction of the bronchial musculature but rather that it was due to a severe congestion of the mucous membrane. He speaks of applying leeches to the chest in cases of bronchitis. He had a definite idea in regard to the differentiation of

asthma caused by bronchial pathology and that caused by heart disease. "When anything makes the heart act inordinately so as to render the function of the valves inefficient, a fit of asthmatic breathing will be the result." He noted them to be more frequent in the old, in which heart diseases were more apt to arise. (23)

He speaks of the end result of valvular diseases - "These cases destroy life by the secondary disease of the lung, the capillaries of the bronchi being kept in a continual state of congestion, incompatible with the function of respiration. The patient becomes dropsical, with livid lips and sinks. The lung itself becomes aedematous. The disease is incurable as there is no means of repairing the valves; it may, however, be palliated for years; and even when the patient appears almost suffocated, relief may be obtained." On his treatment he mentions "Taking a few ounces of blood will restore the freedom of the circulation but caution must be used as it weakens the powers of life." He recommended the dry cupping method of the French for relief. "A great deal may be done by imparting tone to the capillaries of the lung themselves, so as to enable them to resist the regurgitating pressure. (23)

He gave senega in a decoction or infusion given in conjunction with whatever diuretic laxative or other medicine indicated. He understood the modern conception of orthopnea;

the dyspnoea is increased by every attempt to lie down because the heart can not empty itself and of course more blood is thrown upon it in the horizontal than in the erect position. "It is in these cases of asthma that you find other visceral derangements so common, from the same cause - a venous congestion insupportable to the organs. The kidneys, for instance, becoming imperfect in their function, abundantly excreting albumin with a deficiency of the usually secreted salts." It was evident that Billings was very anxious to do away with the "sui generis" idea of disease of the heart and although he did not add a great deal to the development of our present knowledge of "cardiac decompensation", he made an attempt to classify the diseases of the heart. The one thing he did do was to hold quite a modern concept of the all important symptom of dyspnoea and gave an accurate idea as to the method of interpretation from percussion. (23)

The symptom of edema, which had before been suggestive of heart failure to medical men, but never clearly understood, was elucidated by the work of Dr. Bright. In his writings we find the first article written specifically associating a symptom of "failure" complex with the heart. He wrote an article entitled "Anasarca from Heart Disease". He cited a specific case history which we shall quote:

"Girl age 26 admitted to Bartholomew's Hospital the 3rd of March, 1860. Her countenance was pale and sallow;

the skin natural but increased in temperature and dry, during the night the tongue clean and moist; pulse small, thready and feeble, rate 124; thirst excessive, appetite wanting. The inferior extremities are edematous and the body is considerably emaciated. She complains of thoracic pain and cough attended by a frothy pituitous expectoration occasionally streaked with blood. The heart palpitates at the least attempt at exercise. The dyspnoea is so urgent as to require the semi-recumbent position during repose. The abdomen is full and tender to pressure; bowels regular and the secretion from the bladder is scantily voided. She had been subject to cough, but healthy till the last two months, when she became exposed to a cold. She coughed and became dyspnoeic. Symptoms had increased in severity lately. She has never been the subject of rheumatic fever." He noted crepitation in the lungs.

Dr. Bright's work was significant in that he separated the edema caused by kidney lesions and those due to a failing circulation. We find from his writings also that the significance of rheumatic fever in heart disease was well known. We find further that he was well aware of the value of opium in the treatment of the terminal stages of heart disease.

The autopsy performed on the above case was described in a vividly modern way. (24)

In his book on the heart in 1839, Dr. Hope speaks of "cardiac insufficiency" in rather a vague manner by entitling his article "Softening of the Heart." (166) We find in none of the text books or articles written before this time any mention of the whole group of symptoms even suggesting a medical entity. We know they must have considered the terminal event of the heart as did the ancients and although they knew many things which predisposed to early failure of the heart, they were so engrossed in interpreting the meaning of large hearts, irregular hearts, dilating hearts, changes in pulse and at this particular time, sounds produced in the cardiac cycle, that they had not reached a conception of it all which could be drawn into any specific subject. (78) They were, in fact, endeavoring to catch up or interpret the findings which the mechanical aids, the stethoscope, sphygmograph and autopsy findings offered them. (58)

In Trousseau's Medicine" we find they had in many respects a strikingly modern concept of "cardiac failure." They realized the difficulty in prognosticating the length of life in heart affections. They had found, through the singular experiments of Mchuveau and Rouannet upon horses, what caused the abnormal sounds of the heart. (21) We find they associated the seriousness of insufficiencies of the aortic valves to sudden death. (12)

Men spent the next few years in figuring the specific affections of the valves and their final effect upon the heart and circulation. It is at this time we begin to read in the literature of "congestion" and "venostasis." To quote Trousseau: "In addition to the embarrassment of the venous circulation, there is embarrassment of the capillary circulation which declares itself by a livid tint of the skin, swelling of the face, puffiness of the eye lids, a bluish color of the lips and more or less injection of the skin and extremities." He also stated that embarrassed respiration disturbed cerebral function and sanguinous congestion, which might lead to hemorrhage or cirrhosis of the liver and finally edema, anasarca and effusion into the serous cavities. These were all considered phenomena chiefly of a mechanical obstruction of the circulation. He said: "I say chiefly the result of mechanical obstruction because mechanical obstruction is not sufficient in itself to explain the production of the morbid phenomena of which I now speak." (151)

They were becoming aware of the fact that some individuals succumbed rapidly after having presented all the general and rational symptoms of cardiac disease and yet in whom it was never possible during life to recognize well marked local signs of such an affection in the postmortem rooms. Examinations of these bodies found no lesions to explain symptoms at death. (58)

"We likewise see patients presenting all the physical signs of serious disease live for a very long time, and moreover without presenting symptoms of general disturbance of the economy apparently imminent from the passage of the blood through the heart. " (151)

It was well known that an exaggerated susceptibility of the central venous system placed those suffering from disease of the heart in an untoward position. To sum up the status of the conception of "cardiac failure" at this time we turn to a statement of Trousseau speaking of a badly decompensated individual who had been relieved of symptoms but returned a few days later: "If I may so express myself, the cup already too full to the brim, a single drop will cause it to overflow." Dr. Bean said "many of the symptoms of heart sufferers are caused by 'asystole' or want of contractile power. He later called it cachexia of the heart muscle. (151)

We begin to see how the irregularities of the clinical symptoms and the pathological findings were beginning to be a strong impetus to medical men for further investigations into the cause of "cardiac failure". (75) Dr. Bean said: "If we only take into account the anatomical lesion, an organic affection of the heart is not in reality a disease." Although the men were absorbed mainly with congestive types of heart failure, there were men who believed there were causes other than impatent valves, venostasis and congestion.

Dr. Mauriac wrote "We have to estimate in a general manner the causes of death in persons affected with disease of the heart. It is indispensable if one wishes fully to grasp the problem and to look at the question in a manner at once philosophical and medical; to examine, in the first place, the share which certain diathesis have in the production of secondary phenomena of these diseases, when often a longer or shorter period they throw the economy into a peculiar state of cachexia which is conventionally designed "cardiac cachexia." It is known that this special cachexia is the source of profound modification in the crasis of the humors and the two main phenomena are asthenic condition of the circulation and an abnormal exhaltation of serosity into the cellular tissue." (109)

We do not find the text books of the early nineteenth century considering the diseases of the heart in a group. (109) It appears that the subject entered the French text books first. (16) Then the English and following that, the American. (114) The accounts are difficult to trace and although they were certainly conscious of the diseases of the heart and had very modern ideas in many respects, the organization and classification of heart diseases did not appear until later. The early books of Osler, Pepper, Loomis and Thompson in the period of 1883-1889 began to have

a section devoted to "vascular diseases." Cardiac hypertrophy and dilitation were considered as synonomous with "heart insufficiency." (14) To these men at this time anhypertrophied or a dilated heart meant a diseased heart, (14) and one which was low in reserve and apt to fail. We shall consider their ideas of the subject regarding hypertrophy and dilitation somewhat later in the paper.

Loomis and Thompson in the American System of Practical Medicine published in 1889 devoted several pages to the subject of the "embarrassed heart." They list in this volume the causes preventing the work of the heart:

1. Dilitation and congestion.
2. Failure in lobar pneumonia.
3. Relative incompetence. (148)

Pepper's "System of Medicine" published in 1885 had done practically the same thing. Osler (Macrae) "Modern Medicine" in Volume III, Page 719, devoted perhaps the earliest pages to "cardiac insufficiency" in which he gave a practical review of the knowledge of "cardiac failure" as outlined in the preceding pages of this paper but they were under the subjects of carditis and hypertrophy. Strange to say the literature, that is, the current journals, did not consider the subject as "cardiac insufficiency" until the beginning of the nineteenth century. The very first article discovered even in the text books to be labeled specifically "cardiac insufficiency" or any of the other synonyms was

found in Charles F. Hoover's chapter in Osler's "Modern Medicine", (Macrae), Volume IV, Page 187, published in 1907.

The periodicals to which we have had access in the preparation of this paper and which were the first to publish articles relative to "heart insufficiency" are as follows: The French article by Belot, "L'insuffisance Aortique d'Origine Traumatic", 8^o Lyon, 1903, is perhaps the first article, at least listed in "The Surgeon General's Index." The following year, 1904, we find an article written in the Northwestern Medical Journal, II, pages 534 to 536, on "Heart Failure Due to Acute Indigestion." From this time on articles began appearing in most all the journals, both foreign and American. In rather an exhaustive review of the "London Lancet we were not able to find the subject mentioned until after the above years.

Although not important, for a matter of emphasis and general interest, the writer has included the above which almost appears to be a paradox but in the review of the literature this situation was actually found to be the case.

We find from this that our subject at hand is a recent one, a very recent one, yet the actual knowledge had been growing from the time of Hippocrates. In considering the nomenclature which is suitable for the subject, we may say with the myriad of names which has been used to describe the condition

such as "soft heart", "weakened heart", "dilating heart", "cardiac weakness", "failure of hypertrophy", "myocardial insufficiency", "vascular failure", "cardiovascular failure", "congestive failure", "cardiac decompensation", "heart failure", "coronary failure" and many others, it is difficult to determine just which term we might be obliged to use when the heart is not doing its work well. However, we may say that the "Surgeon's General's Index" lists the condition as "cardiac insufficiency" and White's "Heart Disease" published April, 1931, lists it still as "congestive failure and"myocardial insufficiency." We are obliged still, it appears, to use the term in whatever way the circumstances and the apparent etiology of the immediate disaster might call for. (159)

In the period of 1905 the writings upon the subject became very voluminous. Krhel and others were beginning to show that any of the various parts of the organism might be diseased without necessarily disturbing the general circulation. They were just beginning to see the paradox of failure of an organ possessing such a compensating mechanism. (14)

It was shown that the amount of work done by the heart depended upon the size of the ventricular cavity in diastole, the number and intensity of ventricular contractions and the degree of constriction of the blood vessels. (14) The latter in the lesser circulation depending upon the condition of the lungs, whereas in the greater circulation, it depended

upon the condition of the smooth muscle fibers and upon the vasomotor vessels which supply them . The consciousness of the importance of the demands upon the heart leading to failure were at last being somewhat understood. This occurred when larger amounts of blood were needed for food and to keep up the carbon dioxide tension and to overcome the increase in peripheral resistance. (67)

According to Krehl "Not every man's heart is capable of the most extreme exertion." It was becoming evident that weight of the heart bore a definite relation to the skeletal weight, and that the heart muscle improved in function with an improvement of general skeletal muscular efficiency. In times of stress the pathological heart might propel several times the quantity of blood it was used to sending. This power, it was found, expressed itself both in powers of dilatation and increased contraction. (14) Increased work demands that each muscle fiber must contract more quickly, completely and energetically. (142) In the failing heart it was found that the contraction following an increased call upon the heart was, as a rule, unusually powerful and that the elasticity and contractibility of the heart might be increased by venous influences. It was a question and still is, just what part hypertrophy plays in the role of "failure."

Krehl had shown that increased work was made possible only by increased chemical composition and an increased rise in arterial pressure. (14) Thus a vicious cycle was formed, particularly in face of diseased blood vessels. Krehl believed that the muscular insufficiencies around the valvular orifices had a great deal to do with decompensation. (14) Contrary to Buhl and Albrecht, Krehl believed hypertrophy to be due to the response of the myocardium in increased work and not to myocarditis. He also believed that the cause which called for hypertrophy was a factor in damaging the muscle itself. (70)

Although, according to Aschoff, Mortius and Rhomberg, they were able to show that hypertrophy increased also the reserve force, Krehl and others showed that an overworked, functionally impaired and exhausted tissue became a fertile soil for the extension of morbid process and although the hypertrophied heart formed "locuminoris resistant", a limit was finally reached and the primary causes of thrombosis, emboli, acclusions, anemias, fresh inflammations, poisons, and anatomical variances finally interfered with the conduction paths and failure was imminent. (14) It was shown that the heart did not stop from fatigue so readily as from overdistension, according to Lewis. Vagus influences play a great part. Bauer concludes these influences may not only interfere with contractibility but also dilatation. (12) Thus

hypertrophy and dilitation took its debatable place in the causation of cardiac failure and clinicians were beginning to find that the term "myocarditis" was only an excuse for ignorance.

We find in the literature the ever-growing impetus to know why the heart failed and how it might be prevented.

(3) Out of the investigations that had gone before and with the help of the electrocardiograph to study the irregularities of the heart, grew the theory of the causation of the objective symptoms by venostasis and back pressure. (20 - 48) This subject occupied the minds of cardiologists for years and is still an enormous factor to be considered.

According to "Gaskell's Principals" the heart may be said to be inadequate when any of the five essential functions are impaired - rhythmicity, excitability, conductivity, contractibility and tonicity. (161) When we speak of cardiac inadequacy we speak mainly of tonicity so the first result of impaired tonicity is dilitation and it is by the early recognition of dilitation, we recognize that the heart power is no longer equal to its task of maintaining an adequate circulation. (161) The essential features in the picture of mechanical obstruction are due to continuous dyspnoea which is the result of pulmonary stasis, increase in cardiohepatic dullness which proclaims the insufficient evacuation of the cardiac chambers, diminution in the quantity of

urine, which happens as soon as the venous system becomes engorged at the expense of the arterial system. Finally dropsy and cyanosis, which supervene as soon as dilitation exceeds the limit on the frontier between "asystole" and "hyposystole", completes the picture. (67) It seemed that back pressure was certainly the explanation to many forms of heart failure which, without doubt, was true.

Back pressure did not explain the whole picture however, for according to Makenzie as he discussed heart failure in patients with high blood pressure in which there were no demonstrable valve lesions, he came to the following conclusions: "When patients succumb to failure of the heart in consequence of high blood pressure, there is usually some certain degree of change of the heart muscle found. These changes are supposed to be enough to account for the heart failure, yet a careful analysis of the symptoms during life reveals a variety of conditions which can be referred to the peculiar manner in which the heart fails. Generally speaking, the giving away of the heart muscle is described as "heart failure" but this term embraces a great variety of definite forms of heart failure, the recognition of which leads to a more philosophical appreciation of the heart's condition." (98) He believes that the stress laid upon the principals of Gaskell leads us to realize that different functions of the heart are exposed to different and unusual stresses according to the varying circumstances of "modo vivo." (98)

As Harrington said, "There come periods when newer acquisitions are apt to engross the attention to the prejudice of the older possessions." There was a time when men would not but let the back pressure theory explain the whole syndrome and eventual failure of the heart. (67) They argued that heart failure declares itself when under the normal conditions of life an effective circulation ceases to be maintained and that back pressure itself was a consequence of failure and became secondarily an actual cause. There was the doubt, however, just how far the central impediment was the cause of the back pressure. "Its working is as retrograde as the Parthian arrow." (3 - 48) The oncoming blood finally/^{forces}the the central organ to the limit the "vis a tergo" is counterbalanced by the stretch of the tissues. Under the raised pressure the capillaries in particular ooze in excess of the drainage capacity of the lymphatics and the part becomes more or less water-logged. (156) Due to this area of high blood pressure, engorgement, stasis and exudation, function of the part is brought more or less to a standstill and disintegration preponderates the picture of relative death by block. Although collateral channels help keep down the block and increased pressure helps to open the capillaries by a compensatory mechanism, it is not enough to withhold the oncoming amount of blood and the coronary arteries of the heart suffer the same event and finally the "driving power" of the heart ceases. (132)

It is strange that the modern cardiologist would restrict the essential cause of heart failure to a functional impairment of heart muscle. This is about as reasonable as to say that bowel failure can only result from functional impairment of the intestinal musculature. We know that failure of the bowel may be due to obstructions and strangulations and finally defy the healthiest peristalsis and death threatens unless the obstruction is removed. (132) They were convinced at the time that the theory of back pressure had stood the test of time and to belittle its significance was not in keeping with clinical experience, that "argumentum ad aergotantem", which in dealing with the sick man, his griefs and their cure or relief, must ever speak the final word. (132)

Nevertheless as the reports from the autopsy rooms were not coinciding with the clinical diagnoses, great differences of opinion arose as to the etiology, mechanism, prognosis and prevention of "cardiac failure." Albutt said "The jest of death from shortness of breath had vanished but the platitude remains, with a slight modification, we now call it heart failure and the vogue of appendicitis is threatened. Is this a parrot phrase as a kind of shorthand between the doctor and the registrar of death, Or does it, like appendicitis, signify a fresh point of pathology claiming its catchword, Does the phrase mean that a snap of the heart stopped the whole machine as otherwise working on, as a snap of a

connecting rod may stop the engine of the Olympic, Or does it mean that the heart was the ultimem moriens and so long as this central organ held to it, death was kept at bay, In this case, to say that one died from heart failure is surely as empty of truism as to say that one dies from shortness of breath." (3)

In appealing to the clinician, we hear of failure infections, cardiac dilitation, defeat under high pressure, venous or arterial, of senile decay, angina pectoris or aortic regurgitation. The pathologist speaks of fatty degeneration, cardiosclerosis, rheumatic diphtheritic myocarditis, fibroid disease of the heart and snapping of the AV bundle. (3)

Their opinions are decisive and the data enormous, but when asked to draw some parallel between the living patient and the formidable modes of decay, how and when these degradations are manifested, how long the course is to occur before the imminence of death is to be foreseen and provided against, or how we are at times to know any such process is going on at all, the clinician withdraws, as does the pathologist, when the hearts at autopsy do not parallel the clinical findings and symptoms. Thus the consideration of cardiac failure became distinctive, not only with the cardiologist but also the clinician, the pathologist, physiologist and finally the chemist. (3)

In the old days it was enough to say the patient is dead, here is the lesion. But it remained for men like Kirkes, Bence, Jones, Wilks, Fagge, Greene and a host of others to demur, to untangle the vast number of lesions so inconstant, present in one, not present in others, yet death came suddenly, to tell how far it is between the shore of death and the opposite side of beginning loss of reserve of the heart; moreover, just what the raging torrent between these extremes of nervous control, physiology with its endocrine secretions, the tone and change of tone, the disease and the pathology to the tiniest malphigian terminal, or the integrity and function of the heart might be. (3)

Rhomberg, Krehl, Rosenbach all turn for explanation of the paradox of endurance and of deferred crisis in degenerate hearts, not exclusively to the imminent properties of the organ, but also to its nervous endowments, its physiological status, terminal pathological findings, chemical balance and especially to its ganglionic integrity. (14 - 3) Modern men began more and more to think with Makenzie that cardiac failure is a "functional disaster." According to Meltzer and Lytle it is an exhaustion of the reserve force of the heart in the old, in the young a premature exhaustion, and that failure was not sudden but always of gradual and insidious onset, a gradual, unknown sequence of events. (94) Lytle stresses the early recognition of heart failure by the understanding of the earliest objective sensations of which patients

complain. (94)

Elvy calls attention to the fact that pitting edema of the heel, when no other edema is found, is a significant symptom. He mentions also the point that lowered arterial tonus, if uncompensated by more vigorous heart action, may be a cause of edema not primarily cardiac. (57)

Increased rate of the heart has been receiving more attention as a promonitory sign of cardiac overload. The response of increased rate and more efficient systole is brought about by the impulses to the cortex increasing rate, overflowing to the vagus and a loss of tone results. (71)

Other symptoms not included in the ancients' conception are pain in the wrists, (124) indigestion (100) abdominal symptoms (47), pitting edema of the heel (57) and insensible perspiration (31).

To the recent advances in the study of the irregularities of the heart and their effect on "heart failure", much is due to the electrocardiograph. It was adapted from Einthoven's string galvanometer and developed in the short space of time since 1903. In the hands of Lewis, Rothberger, Winterberg, Einthoven and others it has materially aided in increasing our knowledge of the mechanism of the heart beat, both in health and disease. (109)

As men were convinced of the fact that back pressure, hypertrophy and dilitation were not the last words in "heart

failure", modern men have been paying much more attention to the heart muscle itself. (82 - 60) According to Kerr the back pressure theory has done much to direct attention away from the physiological function of the heart muscle. (82) Makenzie had long before advanced the theory of a "rest force which is employed in maintaining an efficient circulation and a reserve force which is called into action when effort is made." (98) In "heart failure" the "reserve force" is the first to suffer. (82)

Robinson believes to understand "heart failure" we must consider the heart as a part of the respiratory system. (127) There is much evidence, as shown by Field and Bach as well as workers in Vienna, that the output of the heart per beat per minute may actually be increased when the heart begins to fail and that the reduction of the output is favorable to the establishment of function. (126 - 60) It has been shown by Blalock, Harrison and others that experimental anoxemias have resulted in augmented minute output of hearts. (70 - 60)

Robinson considers that type of cardiac failure in which there is sudden collapse, small pulse, low pressure, weak rapid sounds, cold extremities and sub-normal temperature as being due to a decrease in output. (128)

During the last few years there has been a great deal of discussion in regard to the difference in points of failure of the heart. White has shown we may have right ventricu-

lar failure without failure of the left ventricle. (162)

The first writings of the modern day have been solely upon an anatomical basis, particularly when conceived as cardiac in origin. The reasons have been that the special senses have been appealed to, the murmurs produced by valvular effects are easily heard, the irregularities of rhythm are recognizable either by palpitation of the pulse or graphic records such as the electrocardiograph. (102)

The rest of the disorders are covered by that all embracing term "myocarditis." The term has probably done more to retard our investigation than any other factor. "The cardiac muscle, the main spring of the circulatory system, has received but little attention. Twenty years ago the stethoscope was the last court of appeal, now the electrocardiograph is the main arbiter." But no matter how meticulously the QRS and T wave deflections are studied, the knowledge obtained is limited and what is more becomes almost delphic in its oracular dogmatism, platitude being piled upon platitude. (103)

One of the strongest reasons for the beginning change in concept of "heart failure" has perhaps been, as the life expectancy rises, we see more individuals who reach the age when vascular and myocardial changes are more prone to occur. The causes probably lie hidden in the earlier periods of life associated with infections, intoxications, nutritional disorders, congenital malformations, heredity and other obscure factors. (82) During life the heart has obviously

failed to function and after death it gives every evidence of being a powerful organ. The heart enlarges because of the work hypothesis. (34)

Since the newer investigations have been carried out in the specific fields of chemistry, physiology, pathology and the clinic, it seems to arrive at a modern conception of "cardiac failure." We should attempt to give a brief review of the concepts in the individual fields.

Heart failure to the chemist is related in purely chemical terms. With edema, lowered blood serum, protein levels and slight ologoproteneinemia, the albumin factor suffers the most. In cases of heart failure the globulin concentration has increased the most and in most cases in a compensatory way. Recent workers believe the change is due to the anorexia of the patients and the insufficient protein intake. These failures have little or no effect upon the success of therapeutic measures. (74) Heart failure, whether accompanied by edema or not, has apparently little influence upon the serum, calcium and phosphate levels or the relation of one to the other. (5) In individual cases, however, the value of these constituents of the serum may play a definite part in the success or failure of therapeutic measures. Although interesting, the findings of the chemist have not yet been of material aid to the practicing clinician. (74)

Some of the most modern work upon the heart has been done by the physiologist. Starling has put forth the following

explanation of "heart failure" resulting from tonicity:

"When the heart is dilated and its tonicity impaired, the diastolic distension may be excessive and the heart does not completely empty itself at each systole, so that there is some residual blood." In response to exertion it might be expected that there would be some increase in output as a result of the increased capacity of the ventricle and the increased effort made by the heart, but the main result is increase in the residual blood and not output. An attempt is made by increase in rate of the heart beat, to supply the increased output called for by exertion. This may, to a certain extent suffice for a time but eventually the heart muscle gives out as a result of working at an excessive speed and the output becomes insufficient for the needs of the body. The right or left side may fail first. (25)

The primary constituents for muscle contraction are liable to forms of organic phosphate called "phosphagen" and "glycogen." The lactic acid production in muscle is proportionate to the amount of muscular energy developed. As yet little is known of phosphagen so the physiologist has confined his attention to a study of the glycogen metabolism as it is an important adjunct in "phosphagen" reactions. The heart contains a high percentage of glycogen which is conserved to a preposterous extent when the skeletal muscle is debilitated by wasting disease. Muscle glycogen reaches a low level, for instance, in diabetes. MacLeod and Prendergast have

shown that during starvation the skeletal muscle glycogen decreases while in the heart it actually increases. It is impossible to reduce the cardiac glycogen by direct energy demands if the supply of oxygen is adequate, according to G. T. Evans. (93)

Lovat and Evans have shown that the oxygen consumption is proportional to the work done and Starling has proven that the oxygen consumption of the heart is determined by the diastolic volume. In other words, under physiological conditions, the oxygen consumption at a given diastolic volume is always the same but has no relation to the systolic volume.

As the heart tires and its functional capacity decreases, its mechanical efficiency is diminished, although the total energy liberated at any given initial length of fiber remains unchanged. The fraction of this energy which can be utilized for performance of work progressively diminishes. To do the same amount of work, the heart, therefore, has to dilate continuously and the work is maintained, constant at an ever increasing cost in total energy. (93)

Miller and Starling have shown in the making of heart-lung preparations, that enormous amounts of glucose were necessary in compensating for insulin deficiency. They found that glucose without insulin did not disappear by combustion, also that insulin and glucose changed only if the cardiac work changed at the same time. In other words, insulin and glucose are necessary for efficient cardiac function which is depend-

ent upon adequate glycogen supplies which are provided by these two substances plus oxygen. In certain instances the heart can be its own storehouse. ⁽¹⁰³⁾ Considering these elements in pathology, Katz and Long have shown that by decreasing the oxygen supply to the heart, there was rapid dilatation and failure, contractions became feebler and conduction slower but recovery was possible unless diastolic standstill had occurred. (80)

G. T. Evans found the striking feature that hearts poisoned with diphtheria toxin might be saved by pulmonary ventilation. When this was not done, the poisoned hearts showed to be lacking in glycogen. Death was due to respiratory failure with progressive anoxemia, producing a conspicuous reduction in cardiac glycogen. He also found that carbon monoxide poisoning caused almost complete obliteration of cardiac glycogen. In simple asphyxia the oxyhemoglobin content might be reduced to 13, 11 or 9 percent but from there on down the glycogen began to diminish to about one-third its former volume. (93)

It has been found in lobar pneumonia, broncho-pneumonia, edema of the lungs and other acute respiratory causes of anoxemia when the oxyhemoglobin is below 75%, serious circulatory symptoms arise and the patient rarely recovers. The same is also true with aviators and mountaineers who go to critical altitudes.

There is an interesting effect produced by inflammation disturbing the coronary anulation in syphilitic myocarditis, hypertension and arteriosclerosis in that the cardiac capacity for work progressively declines. But the most spectacular effects are produced by cyanide poisoning when sudden arrest of oxidation occurs, muscle metabolism immediately ceases. (93)

Katz and Long have shown that although the skeletal muscle may withstand rather large quantities of lactic acid from glycogen combustion, the cardiac muscle is extremely sensitive to it. Not over .072 mgm. percent is tolerated. Therefore the heart is much more susceptible to accumulation H ions and has a much more buffering power. Consequently the heart has a greater intolerance to oxygen debt. Acidosis per se does not interfere with glycogen metabolism. In considering the effect of alkalosis as opposed to the above acidosis upon the heart, its effect, according to Evans, is not directly but rather indirectly through interference with respiratory function. In the specific instance of thyrotoxicosis of the heart, it has been shown by Andrus "that the thyrotoxicosed heart is very sensitive to oxygen withdrawal and that the amplitude is more easily depressed by sodium lactate." It is not possible yet by experimentation to replenish the glycogen stores of thyrotoxicosed heart even by the feeding of carbohydrates. (80)

Cruckshank and Shirvastova found that in diabetic hearts perfused with diabetic blood, they did not utilize blood sugar and that this lost power was restored by the addition of insulin. Thus insulin plays no little role in the maintenance of cardiac glycogen. The difficulty presented to the physiologist is the fact that glycolysis takes place very rapidly after death, therefore they must work in an indirect manner. (103)

Long and Makenzie have shown that there is in failing circulations an increased amount of lactic acid in the human and that with each exacerbation of failing myocardium, the amount goes higher. This may be tolerated for awhile but ultimately, when the limit is reached, the heart ceases. Thus patients with circulatory failure cannot increase their oxygen intake beyond the degree of the impairment. This has been confirmed by Eppinger, Kirsch and Swartz. It is evident from the above experiments that there are three important factors known to the physiologist which lead to cardiac failure proper through cardiac glycogen metabolism. They are oxygen want, thyrotoxicosis and insulin deficiency. (103)

There has been a growing tendency in the pathological field to connect the pathological findings with the physiological function. They give as the main causes of heart failure the following:

In the normal heart fatigue of the muscle does not occur because the heart recuperates during the diastolic pause. With the weakened heart, the muscle receives a poor blood supply, less waste is carried off in the diastolic pause, the heart muscle becomes less efficient and the fatigue is interpreted as the same entity but is due to lack of proper blood supply.

A functional insufficiency may also be a cause of cardiac weakness and consequent failure. Thus a sudden functional change of blood supply to the heart may cause an acute cardiac insufficiency before anatomical changes in muscle have time to develop.

It has been shown by Moritz and Dieten that in severe anemias the enlargement of the heart is due partially to the increased minute volume flow of blood.

The next factor, in weakening of muscle, is the anatomical change in the muscle. A disappearance of muscle fibers and increased connective tissue leads to impaired function, loss of muscle striations and imperfect staining of muscle nuclei. Fatty degenerations and circulatory changes in general play no mean part in the ultimate picture of cardiac failure. It has been shown that hypertrophy and dilatation is usually caused by weakening of the heart muscle, with a lessening of ventricular tone, by an increase of the demands upon the heart.

Another factor favoring the dilatation is increased work of the heart, so notoriously produced by long continued arterial lesions such as hypertension, aortic lesions and mitral insufficiency.

Loss of tone plays an important part. The physical and structural changes in the heart express themselves as factors by long continued strains or long continued weakness, causing long dilatations. (75) Dieten implies that mass thickness of the muscle wall may itself lead to enlargement of the enclosed cavity. Hypertrophy of the heart is still considered compensatory but recent work has shown that many or single chambers may become hypertrophied by special strains.

In summing up the views of the pathologist, we may state:

1. Ordinary compensation is evidenced by absence of cardiac symptoms. (Albrecht)
2. A compensated circulation is not a normal one as the potentialities for failure are present in decreased thoracic space and increased pulmonary circuit.
3. It is evident that in both valvular lesions and myocardial diseases that even though normal blood flow is maintained, changes of importance have taken place in the different parts of the circulatory apparatus. (103)

The pathologist offers as the chief causes of decompensation: progressive lesions, extraneous muscular exercise, dis-

ease of the heart muscle, hypertrophy and changes in the cardiac rhythm, mainly auricular fibrillation. (75)

We come at last to the concept of the clinician - the one who is called upon to continue with his stethoscope, the electrocardiograph, morphine, digitalis and practical experience with patients, to treat and relieve as far as possible the grievances of the cardiac sufferer and prevent as long as possible the ghastly age old "shortness of breath." The average clinician is not aware that there is anything wrong with the heart until the patient consults him because he is having trouble carrying on his natural duties. (3)

The clinician is begging for etiologic diagnosis. The classification of heart disease has been and still is, in a crude state. Chaotic is too strong a word to use regarding it but it is decidedly unsystematic and unsatisfactory. The same is true of nomenclature. No wonder then that there is little uniformity in diagnosis made by the best physicians. What a practitioner wants to know is is there a disease or is it disturbance outside and is the heart ~~competent~~ to carry on its work. (73)

To the clinician, as has been suggested before, "cardiac failure" is a functional disaster. (3) It is obvious that the most successful method of preventing or postponing "cardiac failure" is to prevent the cause or to reduce the severity and shorten the duration of the diseases affecting the heart. (28)

Three questions present themselves to the mind of the modern cardiologist. Does "heart failure" represent an irreversible end stage? Is it a road that leads imminently and quickly to the gate of death or is it the result of changes which the physician may learn to reverse? (28)

"Heart failure" is the first step of a vicious cycle, which once established, tends to progress. It may progress in spite of a diminution of the actual cause and in spite of a life carried on at a low level of activity. (3)

Stokes, a great clinician of Dublin, has said "While diagnosis depends upon the existence and appreciation of certain physical signs, the question of prognosis and treatment depends upon the muscular portions of the heart."

White, in this day, says "While we should emphasize etiology and consider it first and generally foremost. We must not lose sight meanwhile of the other two legs of the tripod of cardiac diagnosis: namely, structural change and functional condition. All together the three elements complete satisfactorily our modern idea of analysis of a cardiac disease." Thus for correct diagnosis, we must consider etiology, pathology and disorder of function.

It has been emphasized by Ashoff, Tawara and Cabot that in most cases "cardiac failure" is not to be explained by anatomically demonstrable lesions. Burwell insists that this is not a fact to be bewailed. It is rather a hopeful thing,

since lesions with an anatomical basis are progressive and always lead to a fatal termination and the problem of treatment becomes quite insoluble. (29)

The conclusion is not entirely unfounded for Zondeck and Fahr have found it possible to relieve certain cases of "heart failure" in myxedematous patients by feeding thyroid extract. (29) Scott and Hermann, Alsman and Wenckeback have shown that in certain cases distressing symptoms of "heart failure" are relieved by the administration of vitamin B. (29) Kutschera and Achenberger in Vienna, have shown a fall of calcium and a diminution of lipoids in the failed muscle. Christian has suggested that "heart failure" may be due to the difficulty of diffusion through enlarged muscle fibers. We hold the hope that some such thing may be found for hypertension and arteriosclerosis. (29)

We may readily see, with the literature full of new chemical and physiological discoveries, new things in etiology such as "heart failure" due to angulation of the spine (44), acute indigestion (100), myxdema(103), hypertension (164), and that pregnancy may be a cause (134), the clinician's attention is directed just now to the various laboratories. (45) He is also calling for a newer and better classification and uniform concept of "heart failure" based on an anatomical, physiological, pathological and etiologic understanding. (45)

Finally we may know the exact mechanism of how the heart, "the best motor known to man" (109), beats sixty times a minute, at least 3600 times an hour, 86,400 times a day. For us, heedful and heedless, does this shuttle of life flit to and fro, for us in tireless periods this pendulum of man's gravitation tells the seconds which will never return. (3)

It has been shown above that the last word has not been spoken in "cardiac failure." It is not a subject void of fruitful investigation but rather ever-living, growing and hopeful. "However, some more refined investigations into the intricacies of cardiac qualities must be instituted in the apparent caprices, clinically speaking, if "heart failure" of man is to be explained, computed and foreseen." (3) "Heart failure" is a very complicated notion, one which must be submitted to no less separating analysis than any other subject undergoes before it becomes a clinical entity. (45)

B I B L I O G R A P H Y

There was found only one article in the literature which had special reference to the history of "cardiac failure" and it was in the French literature. The writer offers the following bibliography as a rather complete review of the early and modern American literature on the subject. No attempt was made to quote all the authors in the preparation of this paper.

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